Tight nuchal cord and neonatal hypovolaemic shock

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SUMMARY Two neonates who went into acute hypovolaemic shock due to a tight nuchal cord were successfully resuscitated. The occurrence of this life threatening complication in two low risk pregnancies emphasises the importance of having staff trained in resuscitation immediately available in the delivery unit.

Cord entanglement occurs in about 20% of all deliveries,[1 2] and is associated with anomalies of fetal heart rate during labour, and perinatal asphyxia.[1 3 4] Recently neonatal anaemia, usually asymptomatic, was reported as a complication of even a loose nuchal cord.[2]

This paper describes two mature infants with life threatening hypovolaemic shock caused by a tight nuchal cord and no other obstetric complications.

Case reports

CASE 1

A baby boy weighing 3330 g was born at term after an uneventful pregnancy. The mother was 36 years old, healthy, normotensive, and nulliparous. Spontaneous labour was followed by spontaneous rupture of membranes and the liqour was clear. Epidural anaesthesia (10 ml 0·25% bupivacaine for six hours) was started during labour and the blood pressure remained stable. The second stage was augmented with oxytocin (4 mU/minute for 50 minutes). The cardiotocograph showed early (type I) decelerations, later irregular (type III), and late (type II) decelerations, but there was no fetal acidosis (the pH of the scalp blood was 7·34). Vaginal delivery was assisted by easy elective vacuum extraction. At birth a single loop of cord was tightly wound round the infant's neck and cord transection was required for further delivery. The Apgar scores were 4 at one and five minutes. The pH of the umbilical artery blood was 7·33.

The infant was extremely hypotonic and pale, with a tachycardia. The peripheral pulses were weak, and there were signs of poor capillary perfusion. There was no oedema or splenohepatomegaly. Five minutes after birth the pH of the infant's blood was 7·05. He was immediately intubated and 50 ml of plasma given through a venous umbilical catheter in the delivery room. Shortly afterwards 75 ml of whole blood was transfused because of the suspicion of acute fetal blood loss. Venous haematocrit before the blood transfusion was 33%. Examination of a peripheral blood smear showed a normochromic normocytic anaemia (haemoglobin concentration 90 g/l) without normoblastosis. A few hours after the blood transfusion the child was extubated and 48 hours later he was transferred to the postnatal ward, from which he was discharged in good health when 6 days old.

In this case there was no fetomaternal blood group incompatibility and the direct Coombs' test was negative. The placenta (700 g) and the umbilical cord (length 42 cm) were normal. Cord insertion was paramarginal without aberrant vessels. Fetal membranes were complete and there were no blood clots on the maternal surface of the placenta. Fetomaternal transfusion was excluded because the Kleihauer test performed six hours after delivery was negative. The infant had no signs of external or internal haemorrhage. Ultrasound and computed tomography scans of the brain and abdominal ultrasound examination yielded normal results.

CASE 2

A baby boy weighing 3010 g was born at a gestational age of 38 weeks. The mother was 29 years old, healthy, normotensive and multiparous. The cervix was softened by extra-amniotic instillation of prostaglandin E_2 gel, and low amniotomy was performed two hours before delivery. The liquor was clear. Epidural anaesthesia (12 ml 0·25% bupivacaine for one hour) was started during labour and blood pressure remained normal. The second stage was augmented with oxytocin (6 mU/minute). Internal monitoring showed early (type I) and late (type II) decelerations. Five minutes before birth the pH of the scalp blood was 7·31. The infant's head was delivered easily by ventouse but the neck was tightly entangled by two loops of cord. Because the nuchal cord could not be reduced early clamping and cutting were necessary for full delivery. Apgar scores were 4 and 6 after one and five minutes, respectively. The umbilical arterial pH was 7·05.

Because of hypotonia and obvious pallor the infant was transferred to the neonatal intensive care

unit. Within the first hour of life rapid deterioration ensued with tachycardia, hypotonia, poor peripheral perfusion, extreme pallor, and continuous grunting. The arterial pulses were undetectable and the blood pressure was 30/15 mm Hg. Because of threatening apnoea the infant was immediately intubated and ventilated, but without improvement. A bolus injection of 30 ml plasma was given, and 60 ml of whole blood was transfused 40 minutes after delivery. Just before the transfusion the baby's arterial pH was 6·9. The extreme acidosis was corrected quickly by restoration of the circulating blood volume 15 minutes after transfusion (pH 7·32); this led to considerable improvement in his general condition. The venous haematocrit was 32% before and 45% after the blood transfusion. Further progress was normal and the baby was discharged in good health when he was 5 days old.

As in the first case, investigations to detect the underlying cause of the acute fetal intrapartum haemorrhage were not helpful. Blood group isoimmunisation was excluded. The placenta (650 g) was complete and there was no retroplacental haematoma. No aberrant or ruptured blood vessels were found in the umbilical cord, which was 50 cm long. The Kleihauer test was negative, excluding fetomaternal haemorrhage. There was no obstetric trauma, and ultrasound examination of the neonate's abdomen and brain and computed tomography scan of his head showed no abnormalities. Thus only fetoplacental haemorrhage due to a tight nuchal cord could explain his acute hypovolaemic shock.

Discussion

When acute fetal blood loss occurs it is usually during labour or delivery.[5] Whatever the underlying cause the clinical picture in the newborn is usually characteristic comprising pallor, tachycardia accompanied by irregular respirations or gasping, weak peripheral pulses, hypotension, and acidaemia. Cyanosis is minimal and the infant's colour is not improved by administration of oxygen. Initially haematocrit and haemoglobin concentrations may be normal (or even misleadingly high) if haemoglobin has been measured in capillary blood samples from an infant in shock owing to peripheral stasis. After equilibration between intravascular and extravascular spaces is reached, however, the expected fall due to the haemodilution and anaemia may show itself.[5]

A peripheral blood smear provides valuable information about the true nature of the anaemia, which is normochromic and normocytic without normoblastosis.[5] These physical and haematological findings are in sharp contrast to those found in neonates with chronic fetal blood loss or perinatal asphyxia whose pallor may be equally severe.

Fetomaternal haemorrhage of sufficient magnitude to result in anaemia at birth was ruled out by the negative Kleihauer test, and internal haemorrhage or iatrogenic blood loss could also be excluded. In the absence of vaginal spotting and after careful examination of the placenta and cord that showed no anomalies that could pinpoint the site of blood loss, fetoplacental haemorrhage due to tight nuchal cord was considered to be the explanation of the condition in both infants.

Cashore and Usher reported neonatal hypovolaemia as a result of tight nuchal coiling.[6] Blood volume determination using I^{125} albumin has shown that this can deprive the infant of up to 20% of its circulating blood volume. In a prospective study on the relation between neonatal anaemia and the presence of either a tight or loose nuchal cord, Shepherd *et al* found that five of 27 (19%) neonates with a tight nuchal cord and four of 30 (13%) with a loose cord were anaemic (haemoglobin concentration less than 132 g/l) within the first 24 hours after birth; no anaemia was found in the patients who served as controls.[2] The authors suggested that the aetiology of the anaemia was cord compression: the blood flow in the thin walled umbilical vein was initially obstructed while the infant's blood continued to return to the placenta through the more muscular umbilical arteries, so that the infant was literally 'pumped out'. Unlike our two patients, these infants were not severely affected by their blood loss.

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